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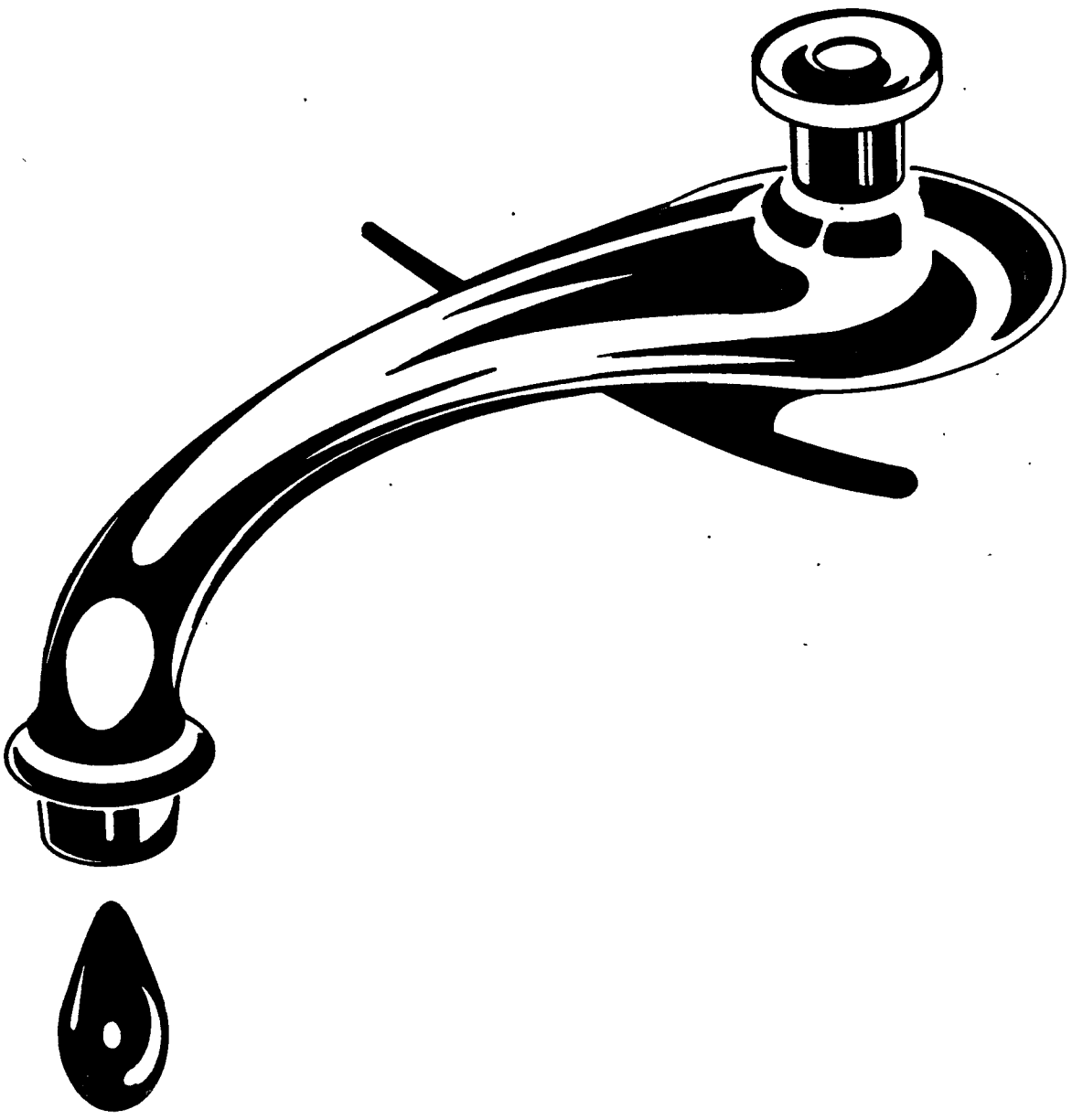
A Benefit Analysis



REDUCING LEAD IN DRINKING WATER:

A BENEFIT ANALYSIS

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SUMMARY

The Safe Drinking Water Act (SDWA), passed by the U.S. Congress in 1974, requires the U.S. Environmental Protection Agency (EPA) to protect public health by setting drinking water standards for public water supplies.* Two levels of protection are described in the SDWA. Primary drinking water regulations, applicable only to public water systems, control contamination that may have an adverse effect on human health by setting either a maximum contaminant level (MCL) or a treatment technique requirement. Secondary drinking water standards are non-enforceable recommendations concerning the aesthetic quality of drinking water, e.g., taste or smell.

The National Primary Drinking Water Regulations (NPDWR) were first promulgated at the end of 1975. EPA revises those regulations by setting maximum contaminant level goals (MCLGs) and related MCLs. MCLGs are non-enforceable health-based goals, intended to protect against known or anticipated adverse health effects, with an adequate margin of safety. MCLs are enforceable limits, to be set as close as feasible to the MCLG; feasibility includes cost and technological constraints. MCLs are proposed at the same time as the MCLGs.

On November 13, 1985, EPA proposed National Primary Drinking Water Regulations (NPDWR) to set MCLGs for 28 synthetic organic chemicals, 11 inorganic chemicals, and 4 microbiological parameters

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* Defined in the Act as water systems serving 25 or more people or having at least 15 service connections.

in drinking water; these substances are listed in Table 1. The proposed MCLGs for probable human carcinogens were set at zero, and MCLGs for other substances were based upon chronic toxicity and other data.

Lead is included among the inorganic substances proposed for regulation in the NPDWR. The current MCL for lead is 50 micrograms of lead per liter of drinking water (ug/l);* the proposed MCLG is 20 ug/l.

The 1986 Amendments to the Safe Drinking Water Act contain a provision banning the use of materials containing lead in public water supplies and in residences connected to public water supplies. States have until June 1988 to begin enforcing this ban.

A. Summary of Study

This analysis estimates some of the benefits that could result from reducing exposure to lead in community drinking water supplies. These benefits are probably much greater than those attributable to just reducing the MCL for lead, but do reflect benefits attainable with reduced exposure to lead through changes in the MCL coupled with changes in EPA's monitoring requirements or other efforts to reduce exposure to lead from drinking water.

There are two primary categories of benefits evaluated in this paper: the public health benefits of reduced lead exposure

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* This is equivalent to and can be stated alternatively as 0.05 milligrams per liter (mg/l), 0.05 micrograms/gram (ug/g), or 50 parts per billion (ppb).

TABLE 1. Substances Included in the 1985 Proposed National Primary Drinking Water Regulations (Maximum Contaminant Level Goals)

A. Synthetic Organic Chemicals

1. Acrylamide
2. Alachlor
3. Aldicarb, Aldicarb sulfoxide and Aldicarb sulfone
4. Carbofuran
5. Chlordane
6. Dibromochloropropane
7. o-,m-Dichlorobenzene
8. cis- and trans-1,2 Dichloroethylenes
9. 1,2-Dichloropropane
10. 2,4-D
11. Epichlorohydrin
12. Ethylbenzene
13. Ethylene dibromide
14. Heptachlor and Heptachlor epoxide
15. Lindane
16. Methoxychlor
17. Monochlorobenzene
18. Polychlorinated biphenyls
19. Pentachlorophenol
20. Styrene
21. Toluene
22. Toxaphene
23. 2,4,5-TP
24. Xylene

B. Inorganic Chemicals

1. Arsenic
2. Asbestos
3. Barium
4. Cadmium
5. Chromium
6. Copper
7. Lead
8. Mercury
9. Nitrate and Nitrite
10. Selenium

C. Microbiological Parameters

1. Total Coliform Bacteria
2. Turbidity
3. Giardia
4. Pathogenic Viruses

and reduced materials damages relating to the phenomenon of lead's presence in drinking water -- as a corrosion by-product. In addition, because the calculation of health benefits depends on the extent of human exposure, another section presents the available data on the occurrence of lead in public water supplies, and presents estimates of the population exposed to drinking water exceeding the proposed MCLG of 20 ug/l. In assessing the benefits of the proposed reduced lead standard, this analysis assumes that EPA will act to reduce lead levels in tap water, as well as maintaining the current high quality of water leaving the treatment plant. It also relies upon and is sensitive to assumptions about drinking water use and consumption patterns.

This analysis estimates the annual benefits for one sample year, 1988, of lowering the amount of lead permitted from 50 ug/l to 20 ug/l. That one year was chosen because environmental lead levels will have stabilized following EPA's 1984 phasedown of lead in gasoline.*

For comparability, all monetary values are expressed in constant 1985 dollars.** The population baseline is the 219.2 million people served by community water systems.

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* Specifically, this analysis measures effects given the conditions on January 1, 1988, when EPA's proposed ban on leaded gasoline will not yet have taken effect. However, even if EPA promulgates that ban, the estimates in this report will not change significantly.

** The 1986 Economic Report of the President to Congress (Table B-4).

1. The Occurrence of Lead in Public Drinking Water

Lead occurs in drinking water primarily as a corrosion by-product; its sources are the materials used in the distribution and residential plumbing systems (cf sources as diverse as the EPA Air Quality Criteria Document for Lead, 1986; Craun and McCabe, 1975; Kuch and Wagner, 1983; Lead in (British) Drinking Water, 1977; etc). Water leaving the water treatment plant is usually relatively lead-free. However, pipes and solder containing lead are corroded by water, and lead levels at the user tap can be much higher than those found at the treatment plant. While the presence of lead service pipes and mains is relatively restricted geographically in the United States, the use of lead solder (and flux) is ubiquitous. And the combination of copper pipes with solder containing lead found in most households can result in high lead levels* in first drawn water that has been in contact with the pipe for a period of time -- levels exceeding the current MCL, even with fairly non-corrosive waters (e.g., Nielson, 1976). In particular, newly-installed solder is easily dissolved, and people living in new housing, or in older housing but with new plumbing, are especially at risk of high levels of lead in the drinking water (Sharrett et al., 1982; Murrell, 1985). In general, lead concentrations in fully flushed water typical of distribution system water, even under corrosive

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* This results from galvanic corrosion, which is the corrosion that occurs when 2 metals, with different electro-chemical potential, are in the same environment.

conditions and with new solder, are generally below 50 ug/l and usually below 20 ug/l.

Because lead occurs generally as a corrosion by-product in U.S. community water supplies, levels in fully-flushed water and in distribution water are typically low. Exposure to lead, however, is from tap water that can contain significant amounts of lead. The estimate of occurrence, therefore, is based upon data collected and analyzed for EPA's Office of Drinking Water in 1979-81. These data portray partly flushed (30 seconds), kitchen tap samples collected by the Culligan water-softening company;* J. Patterson of the Illinois Institute of Technology analyzed the data. Current evidence indicates that these samples are more representative of consumed water than are the fully-flushed samples taken in compliance with EPA's monitoring regulations. The Culligan data indicate that 16 percent of partly flushed water samples could exceed an MCL of 20 ug/l at the kitchen tap. The findings from this data source are consistent with other analyses of the occurrence of lead in tap water and with studies of lead leaching rates in corrosive and non-corrosive waters.

To this must be added the inhabitants of housing built within the past 24 months and that have plumbing materials containing lead. Many studies have shown that new solder can release significant amounts of lead into water, even exceeding the current MCL of 50 ug/l (e.g., Sharrett et al., 1982; Internal Corrosion of Water

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* The use of company names and the presentation of related data does not constitute endorsement of their services.

Distribution Systems, 1985). While corrosive waters have the highest lead levels, relatively non-corrosive waters can also leach significant amounts of lead. The highest lead contamination levels occur with the newest solder (i.e., during the first 24 months following installation), but those levels decline and are generally not elevated beyond five years (e.g., Sharrett et al., 1982; Lassovszky, 1984).

There were 1.7 million new housing starts and permits in the United States during 1983 and 1.8 million in 1984.* Construction data show that housing typically takes six months to a year from permit to potential occupancy, so there are currently about 3.5 million new housing units (i.e., < 24 months). The Statistical Abstract of the United States (1985) indicates that the average household contains 2.73 individuals (Table 58). Multiplied together, a total of 9.6 million people currently live in new housing.

However, not all of these people are served by community water supplies: of the current (1985) U.S. residential population of over 240 million, 219.2 million are served by community water systems and this analysis only addresses that population. In addition, data from the plumbing supply industry show that about 8 percent** of new plumbing is plastic, so 92 percent of the

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* Survey of Current Business, U.S. Department of Commerce - Bureau of Economic Analysis, 1985; Table on New Housing Construction.

** This is the average of claims by the Plastic Pipe Institute presented in Mruk (1984) and of the Copper Development Association presented in Anderson (1984).

population has metal pipes. Therefore, the number of people at risk of high lead levels from new solder in new housing is:

$$9.6 \text{ mil} \times \frac{219 \text{ mil}}{240} \times .92 = 8.1 \text{ million.}$$

To calculate the risk to inhabitants of older housing, subtract the number in new housing (8.8 million)* from the total served by community water systems (219.2 million); that indicates that 210.4 million people live in older homes. Based upon the Culligan data, 16 percent of them (33.7 million) are at-risk of high lead levels from partly flushed water at the kitchen tap. Combining the data from Culligan on lead levels in older housing with the new housing exposure estimates indicated that 41.8 million people using public water supplies currently may be exposed to some water that exceeds the proposed MCL of 20 ug/l; we round this to 42 million.

This may be a low estimate

- because it does not include the potential exposure of occupants in housing built within the past 2-5 years (who also probably remain at greater risk of elevated lead levels);**
- because we have not included those who, while living in older housing, have recently had major plumbing repairs and so are also at risk of the potentially high lead levels associated with newly installed solder;
- because the Culligan data represent water that is harder than average, whereas high lead levels are often found with soft waters; and

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* Derived: $9.6 \text{ mil} \times \frac{219 \text{ mil}}{240} = 8.8$ million people.

** Inhabitants of 2-5 year old housing are not included in this analysis because it was not possible to eliminate them from the base and thus avoid double-counting.

- because the data used are for partially flushed samples, while some people (especially children) may consume water that is closer to first-flush or standing samples (which is more likely to contain higher concentrations of lead).*

In addition, we have not included any data from the estimated 60 million people served wholly or in part by private and non-community water supplies.

There are uncertainties, however, concerning actual patterns of drinking water use and the extent of plastic piping in new construction that would reduce the estimate. Early enforcement of the Safe Drinking Water Act ban on the use of materials containing lead in public water supplies, enforceable in June 1988, could also decrease exposure to lead from drinking water.

The assumptions on the relationship between water lead levels and blood lead levels are taken from the draft (EPA) Water Criteria Document for Lead (1985), which is based upon the recommendations in the Air Quality Criteria Document for Lead (1986). Those documents assume a linear relationship, at least at the lower blood lead levels typical of the United States, with different constants for children and adults. Those formulae are:

(for children)† PbB** = 0.16*** x intake of lead from water

(for adults) PbB** = 0.06*** x intake of lead from water.

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* Water standing in pipes has a greater opportunity for lead to leach into it and, therefore, is more likely to contain higher lead levels.

** PbB = blood lead level

*** These constants have a unit of micrograms-of-lead/deciliter-of-blood per microgram-of-lead-in-water/day, or ug/dl per ug/day.

† This formula was derived from Ryu, 1983. An alternative estimate from the data in that paper suggests a coefficient of about 0.4.

Alternative assumptions (e.g., those reasonably derived from the results of Richards and Moore, 1982 and 1984) could imply that exposure -- and consequently benefits -- may be underestimated, possibly by several factors.

The estimates of the health benefits associated with this proposed rule rely on data on the distribution of blood lead levels in children and adults collected as part of the Second (U.S.) National Health and Nutrition Examination Survey (NHANES II), a 10,000 person representative sample of the U.S. non-institutionalized population, aged 6 months to 74 years. That data base is available from the (U.S.) National Center for Health Statistics and analyses of lead-related data from it have been published before (e.g., Annest et al., 1980 and 1982; Mahaffey et al., 1982; Pirkle and Annest, 1984).

2. Benefits of Reducing Children's Exposure to Lead

Elevated blood-lead levels have long been associated with neurotoxicological effects and many other pathological phenomena: an article on lead's neurotoxicity was published as early as 1839, on anemia in the early 1930s, on kidney damage in 1862, and on impaired reproductive function in 1860. As noted in the Air Quality Criteria Document for Lead (1986), from an historical perspective, lead exposure levels considered acceptable for either occupationally-exposed persons or the general population have been revised downward steadily as more sophisticated bio-

medical techniques have shown formerly-unrecognized biological effects, and as concern has increased regarding the medical and social significance of such effects. In the most recent downward revision of maximum safe levels for children, the Centers for Disease Control (CDC) lowered its definition of lead toxicity to 25 micrograms of lead per deciliter of blood (ug/dl, the standard measure of blood lead level) and 35 ug/dl of free erythrocyte protoporphyrin (FEP). As evaluated in the Criteria Document (1986), the present literature shows biological effects as low as 10 ug/dl (for heme biosynthesis) or 15 ug/dl (for certain renal system effects and neurological alterations); indeed, a threshold has not yet been found for some effects (e.g., elevated levels of a potential neurotoxin* or stature effects, Angle et al., 1982; Schwartz et al., 1986).

There is no convincing evidence that lead has any beneficial biological effect in humans (Expert Committee on Trace Metal Essentiality, 1983; and included in the Criteria Document, 1986).

Elevated blood-lead levels have been linked to a wide range of health effects, with particular concern focusing on young children. These effects range from relatively subtle changes in biochemical measurements at 10 ug/dl and below, to severe retardation and even death at very high levels (80-100 ug/dl). Lead can interfere with blood-forming processes, vitamin D metabolism, kidney function, neurological processes and repro-

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* ALA, or aminolevulinic acid.

ductive functions in both males and females. In addition, the negative impact of lead on cognitive performance (as measured by IQ tests, performance in school, and other means) is generally accepted at moderate-to-high blood-lead levels (30 to 40 ug/dl and above), and several studies also provide evidence for possible attentional and IQ deficits, for instance, at levels as low as 10-15 ug/l. Changes in electroencephalogram readings, as another example, have also been observed at these low levels. For many subtle effects, the data may represent the limits of detectability of biochemical or other changes, and not necessarily actual thresholds for effects.

For children's health effects, two categories of benefits were estimated monetarily: 1) the avoidance of costs for medical care for children exceeding the lead toxicity level set by the Centers for Disease Control (i.e., 25 ug/dl, when combined with FEP levels of > 35 ug/dl) and 2) the averting of costs due to lead-induced cognitive effects. Two alternative methods for valuing the potential cognitive damage resulting from exposure to lead were developed. The first of these two alternatives involves assessing the costs of compensatory education to address some of the manifestations of the cognitive damage caused by lead as a proxy measure for the damage itself. The second relates to one specific indication of that cognitive damage -- potential IQ point loss, and includes a calculation of decreased expected future earnings as a function of IQ point decrement. These estimates neither include many major categories of pathophysiological

effects (e.g., renal damage), nor do either the medical costs or the compensatory education costs consider any lasting damage not reversed by medical treatment or compensatory education. These estimates also attribute few benefits to reducing lead levels in children whose blood lead levels would be below 25 ug/dl even in the absence of the rule.

The estimate of reductions in medical care expenses rely upon published recommendations (Piomelli et al., 1984) for follow-up testing and treatment for children with blood lead levels above 25 ug/dl. The costs of such medical services and treatment were estimated at about $950 per child over 25 ug/dl (1985 dollars). This average reflects both lower costs for most of these children and much higher costs for the smaller subset requiring chelation therapy.

The estimates for compensatory education assumed three years of part-time compensatory education (de la Burde and Choate, 1972 and 1975) for 20 percent of the children above 25 ug/dl, averaging about $2,800 (1985 dollars) per child above that blood lead level based upon data from the U.S. Department of Education (Kakalik et al., 1981).

There is extensive literature examining the relationships between IQ, educational levels attained, demographic variables and earnings (ICF, 1984). The results of that literature were used to estimate the effect of IQ point losses that can occur as a part of the cognitive damage caused by lead exposure upon expected future earnings: one IQ point can directly and indirectly affect earnings by 0.9 percent. The studies of cognitive damage presented in

the Air Quality Criteria Document for Lead (U.S. EPA, 1986) show evidence that blood lead levels of 15-30 ug/dl can be associated with IQ losses of 1-2 points, blood lead levels of 30-50 ug/dl can be associated with IQ losses of 4 points, and over 50 ug/dl of blood lead can correlate with losses of 5 points. Data from the Census Bureau on expected future lifetime earnings, deferred for 20 years* at a 5 percent real discount rate and then annualized, yield estimated benefits of avoided damage from reduced exposure to lead. This alternative method for valuing some of lead's cognitive damage indicated that society could save $1,040 per child brought below 15 ug/dl; $2,600 per child brought below 30 ug/dl; and $2,850 per child brought below 50 ug/dl by reducing lead in drinking water (1985 dollars).

In sum, this analysis indicates that the proposed rule could produce benefits of $27.6 million annually in avoided medical expenses; $81.2 million per year in reduced compensatory education costs; and $268.1 million per year in increased lifetime earnings, based upon sample year 1988; these estimates are in 1985 dollars. Note that compensatory education and affected earnings are alternative methods for valuing aspects of the cognitive damage caused by lead.**

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* These costs are deferred because those suffering the effects are children and will not enter the work force for up to 20 years. Obviously, using the largest deferral period (20 years) reduces the value of the benefit and reduces the benefit estimate, whereas 8- or 10-year-old children may begin working within 8 years and so would have a much shorter deferral period. This biases the estimates downward slightly.

** This also biases the results downward because there is a strong rationale for considering these effects as additive.

In addition, benefits potentially derived by decreasing the incidence of two other categories of health effects (lead's adverse effect upon children's growth and fetal effects) were not estimated in dollar terms. Assuming that pregnant women are distributed proportionately throughout the country, data from the Census Bureau* on birth rates and demographic distributions indicate that 24 percent of the total population is women of child-bearing age (15-44) and that the birth rate is 67.4 births per 1,000 women aged 15-44. Therefore,

41.8 million x 24% x 67.4 per thousand = 680,000.

It is estimated that this proposed rule could prevent 680,000 fetuses from being exposed to elevated lead levels. The fetal effects are particularly important, because several recent studies have shown that lead exposure within the normal range (6-20 ug/l) can be associated with various negative pregnancy outcomes (such as early membrane rupture and even miscarriages, e.g., Moore, 1982; Wibberly et al., 1977), and with low birth weight, inhibited postnatal growth and development (e.g., Bornschein, 1986; Bellinger, 1985 and 1986; Dietrich et al., 1986). In addition, this proposed rule could prevent 82,000 children from risk of growth effects.

3. Blood-Pressure-Related Benefits and Other Adult Health Effects

Lead has long been associated with elevated blood pressure, but until recently most of the studies have focused only on hypertension and relatively high lead levels typically found only in those occupationally exposed to lead. Several recent studies, however

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* Statistical Abstracts (1986), Tables 27 and 82.

(e.g., Pirkle et al., 1985; Harlan et al., 1985; Pocock, 1984 and 1985), have found a continuous relationship between blood lead and blood pressure. These studies provide evidence for a small (compared to other risk factors) but robust relationship after controlling for numerous other factors known to be associated with blood pressure. Experimental animal studies in several species of rats and pigeons also provide evidence of a relationship between moderate blood-lead levels and increases in blood pressure.

To calculate these benefits, logistic regression equations were used to predict how reducing exposure to lead in drinking water would affect the number of hypertensives in the U.S. population. These estimates cover only males aged 40 to 59, because the effect of lead on blood pressure appears to be stronger for men and because the correlation between blood pressure and age is much smaller in this age range, reducing the potential for confounding due to the correlation between blood lead and age. The estimates rely upon 1) site-adjusted coefficients from analyses of the NHANES II data relating blood lead levels to increases in blood pressure* and 2) coefficients relating blood pressure increases to more serious cardiovascular disease outcomes, based on data from the Framingham Study (McGee et al., 1976) and Pooling Project (1976), as confirmed by Levy et al., 1984.

Levy has demonstrated that the risk coefficients from the Framingham Heart Study, when coupled with the observed reductions in blood pressure, smoking, and cholesterol in the U.S. population

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* The specific coefficients and the basis for their derivation are described in the Addendum to the Criteria Document, 1986, which is included in volume 1 of that publication.

during the 1970s, correctly predicts the observed reductions in cardiovascular mortality in the overall population during that decade. The Pooling Project showed that the Framingham coefficients adequately predicted cardiovascular outcomes (such as strokes and heart attacks) in the other five large prospective heart studies performed in the U.S. Therefore, while caution is clearly warranted in view of the limited data on the effect of lowering blood lead levels on blood pressure, use of the regression coefficients from the Framingham Study provide a reasonable basis by which to predict potential changes in cardiovascular outcomes associated with blood pressure changes due to decreased lead exposure.

Based upon this information, reducing exposure to lead from drinking water in 1988 could reduce the number of male hypertensives (aged 40 to 59) by 130,000. Using estimates of the costs of medical care, medication, and lost wages, such a reduction in hypertension incidence would yield a value of $250 per year per case avoided (1985 dollars).

These estimates of how blood pressure reductions would affect the incidences of various cardiovascular diseases were based on projections of changes in blood pressure as a result of the proposed rule and estimates of the relationships between blood pressure and heart attacks, strokes, and deaths from all causes. As noted earlier, the latter estimates were derived from several large epidemiological studies, primarily the Framingham study. However, because those studies included very few nonwhites, the estimates were further restricted to white males, aged 40 to 59. Thus, the benefits estimates do not include middle-aged, nonwhite males.

The basis of most of the medical costs are the cost-of-illness estimates presented in Hartunian et al., 1981, which were adjusted in three ways to reflect current conditions. First, we inflated them to 1985 dollars using data from the 1986 Economic Report of the President to Congress. Second, we adjusted the costs to reflect changes and improvements in medical treatment, including the tripling in the incidence rate of coronary bypass operations that occurred between 1975 and 1982. Third, Hartunian used a 6 percent real discount rate to present-value future expenditures, while this analysis uses a 10 percent real discount rate.

The value of reductions in heart attacks and strokes was based on the cost of medical care and lost wages for nonfatal cases. Expected fatalities from heart attacks and strokes were included in the estimate of deaths from all causes. That procedure yielded benefits of $65,000 per heart attack avoided and $48,000 per stroke avoided (1985 dollars) for the 240 heart attacks and 80 strokes estimated to likely be avoided in 1988 because of this proposed rule. It is important to note that these estimates do not account for any reductions in the quality of life for the victims of heart attacks and strokes (e.g., the partial paralysis that afflicts many stroke victims).

Valuing reductions in the risk of death is difficult and controversial, with a wide range of estimates in the literature. EPA's policy guidelines (U.S. EPA, 1984), for example, suggest a range of $400,000 to $7 million per statistical life saved. Using $1 million per case, the benefits of reduced mortality dominate our estimates of total blood-pressure-related benefits; these total

$240 million in 1988 for the 240 deaths estimated as likely to be avoided in that year. Altogether, the monetized benefits of reducing adult male exposure to lead in drinking water are estimated to total $291.9 million per year (in 1985 dollars), using 1988 as a sample year.

In addition, because lead crosses the placental barrier and is a fetotoxin, pregnant women exposed to lead are at risk of complications in their pregnancies and damage to the fetus. (Fetal effects are discussed above, under children's health effects.) While we have not monetized any of these reproductive effects, as noted above, 680,000 pregnant women per year probably receive water that exceeds the proposed standard of 20 ug/l, and would benefit from the proposed rule. Lead-induced effects on male reproductive functions have also been discussed in the scientific literature but are not included in this report.

4. Benefits of Reduced Materials Damage

A third category of monetized benefits relates to the phenomenon of lead's presence in drinking water: it is a product of the corrosive action of water upon the materials of the distribution and residential plumbing. For the most part, therefore, treatment processes used to reduce high levels of lead in drinking water are the same as treatment processes used to reduce the corrosion potential of the water. Reducing corrosion damage will produce substantial benefits to water utilities, their rate-paying customers, and building owners.

Published estimates of the costs of corrosion damage range from $12 to $46 per person per year (1985 dollars), and are summarized in Table 2. Estimates of the costs that can be avoided by corrosion control measures range from 20-50 percent of total damage. The point estimate of avoidable corrosion costs (i.e., the benefits of corrosion control) is $8.50 per capita annually (1985 dollars). For comparison, estimates of average corrosion treatment costs range from under $1 per person per year (based upon the experience in Boston and Seattle, cities currently treating their highly corrosive waters) to almost $5 per person per year (based upon the highest treatment costs presented in the ODW cost report).* As a point estimate, we assumed per capita annual treatment costs of $3.80 (1985 dollars).

Estimates of the extent of corrosive water also vary. A commonly accepted profile is that developed by the U.S. Geological Survey in the early 1960s, which identified the Northeast, Southeast, and Northwest sections of the country as having the softest and most corrosive waters (Durfor and Becker, 1964a and 1964b). The combined populations of those states are 67.7 million people (1980 census). Assuming that these areas are served proportionately by community water systems,** 61.8 million people

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* The range, however, is quite wide and highly sensitive to system size. These represent average costs. In some very small systems (i.e., serving 25-100 people), costs may be many times higher

** Of the total population of about 240 million, 219.2 million people are served by community water systems.

TABLE 2. Estimates of Annual Per Capita Corrosion Damage (1985 dollars)

Studies	Estimated Annual Corrosion Damage (per capita)			Corrosion Damage Avoidable Through Water Treatment	Annual Per Capita Benefits of Corrosion Control	Assumptions/Notes
	Distribution Systems	Residential	Total			
Kennedy Engineers (1973)	$5.57	—	$16.71*	30%*	$5.01*	Assumed 30% potential reduction in corrosion damage and that distribution costs were one-third of total costs.
Hudson & Gilcreas (1976)	$8.68*	—	$26.04*	50%	$13.02*	They did not include increased operating costs. Per capita estimate assumes 200 million people are served by public water systems. Assumed that distribution costs were one-third of total costs.
Kennedy Engineers (1978)	--	$30.87*	$46.30*	20%	$9.26*	They calculated $6.17 per capita in savings to residence owners. Assumed residential costs were two-thirds of total costs.
Bennett et al. (1979) (cited in Ryder, 1980)	$9.40	—	$28.20*	20%	$5.64*	Assumed that 200 million people are served by public water systems and that distribution costs were one-third of total costs.
Energy & Environmental Analysis (1979)	$3.98	$7.97	$11.95	38%	$4.54	This is an admitted underestimate: it includes only damage to pipes (not damage to water heaters, increased operating costs, etc.)
Ryder (1980)	$1.17	$22.19	$23.36	25%	$5.84	Ryder ascribed 95% of corrosion damage to private owners.
Kirmeyer & Logsdon (1983)	--	$23.60*	$35.40*	40%	$14.16*	Assumed residential costs were two-thirds of total damage.
					AVERAGE $8.21 W/OUT EEA $8.82	

* These estimates have been calculated by the authors of this paper. Assumptions are noted above.

would benefit from actions to reduce the corrosivity of their water. That figure, multiplied by $8.50 per person, yields annual benefits from reduced corrosivity of $525.3 million (1985 dollars).

5. Summary of Benefits of Reduced Lead in Drinking Water

This analysis of the benefits of reducing exposure to lead in drinking water indicates that the monetized annual benefits could range from $926.0 to $1,112.9 million (1985 dollars) for sample year 1988. In addition, there are numerous health benefits of reduced exposure to lead that are not monetized. The annual monetized benefits are summarized in Table 3, and the non-monetized benefits are presented in Table 4.

Based upon the latest cost estimates used by the Office of Drinking Water* the projected benefits exceed the costs by about 4:1. Expressed differently, lowering the MCL to 20 ug/l could produce annual net benefits of about $800 million in 1988.

It should be emphasized that considerable uncertainty is associated with these estimated benefits, uncertainties derived both from the current state of knowledge concerning lead health effects and the valuation of avoiding such effects. Other analogous efforts to estimate benefits associated with reducing lead in drinking water may be useful in helping to judge how reasonable these present benefit estimates are.

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* These calculations use preliminary EPA Office of Drinking Water cost estimates. Costs and net benefits will be discussed more extensively in other documents associated with this proposed rulemaking.

TABLE 3. Summary of Estimated Annual Monetized Benefits of Reducing Exposure to Lead from 50 ug/l to 20 ug/l (1985 dollars) for Sample Year 1988

Estimated population exposed to drinking water exceeding proposed MCL		42 million*
Children's health benefits		
-reduced medical costs		$27.6 million
-reduced costs of cognitive damage		
Method 1 - compensatory education		$81.2 million
Method 2 - decreased future earnings		$268.1 million
TOTAL:	Method 1	$108.8 million
	Method 2	$295.7 million
Adult health benefits (males only)		
-reduced hypertension savings (males, aged 40-59)		$32.5 million
-savings from fewer heart attacks (white males, aged 40-59)		$15.6 million
-savings from fewer strokes (white males, aged 40-59)		$3.8 million
-savings from fewer deaths (white males, aged 40-59)		$240.0 million
TOTAL:		$291.9 million
Materials benefits		
-benefits of reduced corrosion damage		$525.3 million
TOTAL ANNUAL MONETIZED BENEFITS		
-Method 1 - using compensatory education		$926.0 million
-Method 2 - using decreased future earnings		$1,112.9 million
ESTIMATED ANNUAL COSTS		$230.0 million
NET ANNUAL MONETIZED BENEFITS	about	$800 million

* Total population served by community water systems: 219 million

TABLE 4. Summary of Estimated Annual Non-monetized Benefits of Reducing Exposure to Lead from 50 ug/l to 20 ug/l for Sample Year 1988

	Reductions in Numbers of People at Risk
Estimated population exposed to drinking water exceeding proposed MCL	42 million*
Children's health benefits	
- children requiring medical treatment	29,000
- loss of 1-2 IQ points	230,000
4 IQ points	11,000
5 IQ points	100
- children requiring compensatory education	29,000
- children at risk of stature decrement	82,000
- fetuses at risk	680,000
- increased risk of hematological effects	82,400
Adult health benefits	
-cases of hypertension (males, aged 40-59)	130,000
-heart attacks (white males, aged 40-59)	240
-strokes (white males, aged 40-59)	80
-deaths (white males, aged 40-59)	240
-(reduced risk to pregnant women ((women, aged 15-44) (same as fetuses	680,000)))
-reduced risk of reproductive effects (women, aged 15-44)	34,000

* Total population served by community water systems: 219 million

B. Boston Case Study

In the spring of 1986, Jonathan Jacobson analyzed the incremental costs and benefits to the City of Boston of reducing the lead MCL from 50 ug/l to 10 ug/l.* That analysis, carried out as a masters thesis project at Harvard University, focused on Boston as a city with high potential for increased lead exposure via drinking water.

Boston's water is highly corrosive: it is relatively acidic (pH = 6.7) and soft (14 mg/l of $CaCO_3$), and has low alkalinity (Karalekas et al., 1975). Boston also has a large percentage of lead pipes in service. During the 1970s, several studies found high lead levels in Boston's drinking water (e.g., Karalekas et al., 1975; and several internal studies conducted by the Massachusetts Water Resources Authority and the [Boston] Metropolitan District Commission).

To reduce the high lead levels, Boston began corrosion control treatment. Monitoring performed by EPA's Region I from 1975 to 1981 indicated that lead, iron and copper levels dropped significantly (Karalekas et al., 1982). However, while lead concentrations generally decreased to below the current MCL of 50 ug/l, additional treatment will be necessary to comply with a lowered MCL.

Jacobson analyzed the incremental annual costs and benefits of additional efforts by Boston to control further the corrosivity

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* He assumed a lowered MCL of 10 ug/l because that is the feasibility limit for current treatment and technology.

of its water, using sample years 1988 and 1992, and estimating all costs in 1985 dollars. His analysis assumed the following:

- compliance will be measured by a standing "grab" sample, that is, a sample taken immediately after turning on the faucet at any random time during the day after an unknown period of standing;
- it will be impossible for every tap to meet the lowered MCL, even using state-of-the-art treatment, and so samples should be averaged; and
- that, while the effectiveness of specific treatment procedures varies in not-yet-well-understood ways when actually used in the field, corrosion control is ultimately feasible with current state-of-the-art methods.

Jacobson, using data from EPA Region I and the Massachusetts Water Resources Authority, calculated the benefits of additional 2-stage treatment for Boston's water: further raising the pH (to reduce the acidity of the water) and installing several pumping stations to maintain a consistent concentration of sodium hydroxide throughout the system.

Jacobson used the same categories of monetized health benefits* as those described in this EPA analysis, except that he did not include the estimates of cognitive damage associated with decreased future earnings. His estimates of materials benefits rely only upon the Kennedy Engineers (1978) study and information in the American Water Works Association Corrosion Manual (1985).

His results, summarized in pages 36-39 of his study, indicate incremental costs of $700,000 per year (using sample year 1988) and incremental annual benefits of $7.9 million (including estimated

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* For blood-pressure-related estimates, however, he used the non-site-adjusted coefficients from the NHANES II contained in Pirkle et al., 1985.

health benefits of $6.9 million and materials benefits of $950,000; based upon sample year 1988). This yields estimated net annual benefits of $7.2 million and a benefit to cost ratio of 11:1 (compared to the estimate of 4:1 in this analysis).

It is unclear, however, whether or how these results can be extrapolated to other U.S. water systems and cities, and therefore, to this proposed rule.

REFERENCES

American Water Works Association. Water Quality Division Committee report. Determining internal corrosion potential in water supply systems. JAWWA 1984 August; p. 85-88.

American Water Works Association. Internal corrosion of water distribution systems. Comparative research report. 1985.

Anderson R, Berry D. Regulating corrosive water. Water Resources Research. 1981 December; 17(6): 1571-1577.

Angle CR, McIntire MS. Children, the barometer of environmental lead. In: Barness L (ed): Advances in Pediatrics, Yearbook Medical Publishers, Inc., Chicago 1982; pp 3-32.

Angle CR, McIntire MS, Swanson MS, Stohs SJ. Erythrocyte nucleotides in children - increased blood lead and cytidine triphosphate. Pediatric Research 1982; 16: 331-4.

Annest JL, Mahaffey KR, Cos DH, et al. Blood lead levels for persons 6 months - 74 years of age: United States 1976-1980. National Center for Health Statistics, Advanced Data no. 79, May 12, 1982.

Annest JL, Pirkle JL, Makuc D, Neese JW, Bayse DD, Kovar MG. Chronological trends in blood lead levels between 1976 and 1980. New England Journal of Medicine 1983; 308: 1373-7.

Bellinger D, Needleman H, Leviton A, Waternaux C, Rabinowitz M, Nichols M. Early sensory-motor development and prenatal exposure to lead. Neurobehavioral Toxicology and Teratology 1984; 6:387-402.

Bellinger D, Leviton A, Waternaux C, Needleman H, Rabinowitz M. A longitudinal study of the developmental toxicity of low-level lead exposure in the prenatal and early postnatal periods. Paper presented at the Heavy Metals in the Environment Conference, Athens, Greece. 1985.

Bellinger D, et al. Correlates of low-level lead exposure in urban children at 2 years of age. Pediatrics 1986 - in press.

Bornschein RL. "Effects of prenatal and postnatal lead exposure on fetal maturation and postnatal growth", to be presented at the Joint EPA-EEC conference on Lead and Neurotoxicity, Edinburgh, Scotland. September 1986.

Britton A, Richards WN. Factors influencing plumbosolvency. Journal of the Institute of Water Engineers and Scientists. 1981 July.

Comstock GW. Water quality and cardiovascular disease: a review of recent studies in Canada and the US. Paper presented at the International Symposium on Geochemistry and Health, London, England; April 1985.

Craun GF, McCabe LG. Problems associated with metals in drinking water. JAWWA 1975 November; 67(11):593.

Dangel RA. Study of corrosion products in the Seattle water department distribution system, U.S. EPA report 670/2-75-036. Cincinnati, Ohio. 1975.

De la Burde B, Choate MS Jr. Does asymptomatic lead exposure in children have latent sequelae. Journal of Pediatrics 1972; 81: 1088-91.

De la Burde B, Choate MS Jr. Early asymptomatic lead exposure and development at school age. Journal of Pediatrics 1975; 87: 638-42.

Dietrich KN, Kraft KM, Shukla R, Bornschein RL, Succop PA. The neurobehavioral effects of prenatal and early postnatal lead exposure. In Schroeder (ed): Mental Retardation, Neurobehavioral Toxicology and Teratology, 1986 - in press.

Durfor CN, Becker E. Chemical quality of public water supplies of the U.S. and Puerto Rico, 1962. Hydrologic Investigations Atlas HA-200. U.S. Geological Survey. 1964a.

Durfor CN, Becker E. Public water supplies of the 100 largest cities in the U.S., 1962. Geological Survey Water-Supply Paper 1812. U.S. GPO, 1964b.

Energy and Environmental Analysis. Health and corrosion impact of soft water. Prepared for the A/C Pipe Producers Association. 1979 August.

Erickson MM, Poklis A, Gantner GE, Dickinson AW, Hillman LS. Tissue mineral levels in victims of sudden infant death syndrome. I. toxic metals - lead and cadmium. Pediatric Research 1983; 17: 784-99.

Harlan WR, Landis JR, Schmouder RL, et al. Relationship of blood lead and blood pressure in the adolescent and adult U.S. population. Journal of the American Medical Association, 1985 January 25.

Hartunian NS, Smart CN, Thompson MS. The Incidence and Economic Costs of Major Health Impairments. Lexington, MA: Lexington Books 1981.

Hudson HE Jr, Gilcreas FW. Health and economic aspects of water hardness and corrosiveness. JAWWA 1976; 68: 201-204.

ICF. A survey of the literature regarding the relationship between measures of IQ and income. Prepared for U.S. EPA, Office of Policy Analysis, 1984.

Internal Corrosion of Water Distribution Systems. Report of the AWWA Research Foundation and the DVGW - Forschungstelle. 1985.

Jacobson J. The costs and benefits of reducing lead in Boston's water. Masters thesis submitted to Harvard University, Kennedy School of Government, Spring 1986.

Kakalik JS, et al. The Cost of Special Education. Rand Corporation Report N-1791-ED, 1981.

Karalekas PC, Craun GF, Hammonds AF, Ryan CR, Worth DJ. Lead and other trace metals in drinking water in the Boston metropolitan area. Proceedings of the 1975 AWWA Annual Conference, Minneapolis, MN.

Karalekas PC, Ryan CR, Larson CD, Taylor FB. Alternative methods for controlling the corrosion of lead pipe. Journal of the New England Water Works Association 1978 June.

Karalekas PC, Ryan CR, Taylor FB. Control of lead pipe corrosion in the Boston metropolitan area. Proceedings of the 1982 AWWA Annual Conference, Miami Beach, Florida.

Karalekas PC, Ryan CR, Taylor FB. Control of lead, copper, and iron pipe corrosion in Boston, JAWWA. 1983 February.

Karalekas PC. Impact of lead piping and fittings on drinking water quality. Proceedings of a Seminar on Plumbing Materials and Drinking Water Quality. Sponsored by US EPA; Cincinnati, Ohio; 1984 May.

Kennedy Engineers. Internal corrosion study, summary report. Conducted for the city of Seattle, Washington; 1978 February.

Kirmeyer GJ, Logsdon GS. Principles of internal corrosion and corrosion monitoring. JAWWA 1983 February; pp. 78-83.

Kuch A, Wagner I. A mass transfer model to describe lead concentrations in drinking water. Water Research 1983; 17 (10): 1303-1307.

Lassovszky P. Effect on water quality from lead and nonlead solders in piping. Heating/Piping/Air Conditioning, 1984 October; pp. 51-58.

Lead in Drinking Water: A Survey in Great Britain. Pollution Paper 12. Dept. of the Environment HMSO, London, 1977.

Levy RI. The decline in coronary heart disease mortality: Status and perspective on the role of cholesterol. American Journal of Cardiology 1984 August 27; 54 (4).

Mahaffey KR, Annest JL, Roberts J, Murphy MS. National estimation of blood lead levels: United States (1976-1980). New England Journal of Medicine 1982; 307: 573-9.

McGee D, Gordon T. The results of the Framingham Study applied to four other U.S. - based epidemeologic studies of coronary heart disease. The Farmingham Study. Section 31. DHEW Pub No. (NIH) 76-1083. National Institutes of Health, Washington, D.C. U.S. Government Printing Office 1976.

Midwest Research Institute. Occurrence and impacts of aggressive water systems. MRI publication. 1979.

Millette JR, Hammonds AF, Pansing MF, Hanson EC, Clark PJ. Aggressive water: assessing the extent of the problem. JAWWA 1980; 72 (5): 262-66.

Moore MR, et al. Some studies of maternal and infant lead exposure in Glascow. Scotish Medical Journal 1982; 27: 113-122.

Mruksa. Plastic piping and joining materials. Presented at: Plumbing Materials and Drinking Water Quality Seminar; sponsored by EPA; Cincinnati, Ohio; 1984.

Multiple Risk Factor Intervention Trial: Risk factor changes and mortality results. Multiple Risk Factor Intervention Trial Research Group. JAMA 1982; 248: 1465-77.

Murrell NE. Impact of metallic solders on water quality. Presented at the Specialty Conference on Environmental Engineering, American Society of Corrosion Engineers. Boston 1985.

National Center for Health Statistics. Plan and Operation of the Second National Health and Nutrition Examination Survey 1976-1980. National Center for Health Statistics 1981 (Vital and Health Statistics Series 1, No. 15).

Nielsen K. Contamination of drinking water by cadmium and lead from fittings. Danish Building Research Institute. 1975.

Nielsen K. Dissolution of materials from service pipes and house installations and its sanitary aspects. International Water Supply Association. Amsterdam Conference; 1976.

Nielsen K. Corrosion of soldered and brazed joints in tap water. British Corrosion Journal 1984; 19(2): 57-63.

Oliphant R. Lead contamination of potable water arising from soldered joints. Water Research Centre Report #125E. England; 1983.

Patterson JW, O'Brien JE. Control of lead corrosion. JAWWA 1979 May; 71(5): 264-271.

Patterson JW. Corrosion in water distribution systems. Prepared for U.S. EPA, Office of Drinking Water. 1981 March.

Piomelli S, Seaman C, Zullow D, Curran A, Davidow B. Metabolic evidence of lead toxicity in "normal" urban children. Clinic Res 1977; 25: 459A.

Piomelli S, Seaman C, Zullow D, Curran A, Davidow B. Threshold for lead damage to heme synthesis in urban children. Proceeding of the National Academy of Sciences in 1982 May; 79: 3335-9.

Piomelli S, Rosen JF, Chisolm JJ, Graef JW. Management of childhood lead poisoning. Journal of Pediatrics 1984; 4:105.

Pirkle JL, Annest JL. Blood lead levels 1976-1980 - Reply (letter). N Eng J Med 1984; 310(17): 1125-6.

Pirkle JL, Schwartz J, Landis JR, Harlan WR. The relationship between blood lead levels and blood pressure and its cardiovascular risk implications. American Journal of Epidemiology, 1985; 121(2): 246-58.

Pocock SJ, Shaper AG, Ashby D, Delves T, Whitehead TP. Blood lead concentration, blood pressure, and renal function. British Medical Journal 1984; 289: 872-874.

Pocock SJ, Shaper AG, Ashby D, Delves T. Blood lead and blood pressure in middle-aged men. In: Lekkas TD (ed), International Conference on Heavy Metals in the Environment. September 1985; Athens, Greece.

The Pooling Project Research Group. Relationship of blood pressure, serum cholesterol, smoking habit, relative weight and ECG abnormalities to incidence of major coronary events: Final report of the pooling project. J Chron Dis 1978; 31: 201-306.

Provenzano G. The social costs of excessive lead-exposure during childhood. HL Needleman ED. Low Level Lead Exposure: The Clinical Implications of Current Research. New York: Raven Press 1980.

Public Use Data Tape Documentation, Hematology and Biochemistry. Second National Health and Nutrition Examination Survey 1976-1980. Catalog number 5411, U.S. Public Health Service, National Center for Health Statistics.

Richards WN, Moore MR. Plumbosolvency in Scotland - the problem, remedial action taken and health benefits observed. JAWWA 1982 May; p. 901-918.

Richards WN, Moore MR. Lead hazard controlled in Scotish water systems. JAWWA 1984 August; p. 60-67.

Ryder RR. The costs of internal corrosion in water systems. JAWWA 1980 May; pp. 267-279.

Ryu JE, Ziegler EE, Nelson SE, Fomon SJ. Dietary intake of lead and blood lead concentration in early infancy. Am. J. Dis. Child. 1983; 137:886-891.

Schwartz J, Angle C, Pitcher H. The relationship between childhood blood lead levels and stature. Pediatrics 1986; 77: 281-288.

Schwartz J, Leggett J, Ostro B, Pitcher H, Levin R. Costs and benefits of reducing lead in gasoline. U.S. EPA, Office of Policy Analysis 1985.

Sharrett AR, Carter AP, Orheim RM, Feinleib M. Daily intake of lead, cadmium, copper and zinc from drinking water: the Seattle study of trace metal exposure. Environmental Research 1982; p. 456-475.

U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office. Air Quality Criteria for Lead. 1986 March.

U.S. Environmental Protection Agency. Regulatory Impact Analysis Guidelines. 1984.

Wibberly DG, Khera AK, Edwards JH, Rushton DI. Lead levels in human placentae from normal and malformed births. Journal of Medical Genetics 1977; 14: 339.